Claims as Pending in U.S. Appln. No. 09/321,247, Upon Entry of Amendment

Responsive to Office Action mailed July 17, 2001 (Paper No. 11)

1. An expression vector, wherein the expression region comprises:

a promoter;

an intracellular retention signal sequence encoding region, and

a chemokine encoding gene;

wherein said intracellular retention signal sequence and said chemokine encoding gene are expressed from said promoter as a single intrakine transcript.

2. The expression vector of claim 1, further comprising a gene encoding a secreted chemokine.

3. The expression vector of claim 2, wherein said gene encoding said secreted chemokine is expressed from an internal ribosome entry site.

4. The expression vector of claim 1, further defined as a retroviral vector.

5. The expression vector of claim 1, wherein said intracellular retention signal sequence is an endoplasmic reticulum retention signal sequence.

6. (Amended) The expression vector of claim 5, wherein said endoplasmic reticulum retention signal sequence is a KDEL sequence (SEQ ID NO:7).

7. (Amended) The expression vector of claim 6, wherein said KDEL sequence (SEQ ID NO:7) has the amino acid sequence SEKDEL, SEQ ID NO:6.

8. (Amended) The expression vector of claim 1, wherein said chemokine gene encodes a chemokine that binds to a C-C chemokine 5 receptor, a C-C chemokine 3 receptor, a C-C chemokine 1 receptor or a CXR4 receptor.

9. (Amended) The expression vector of claim 1, wherein said chemokine gene encodes a chemokine that binds to a C-C chemokine 5 receptor.

10. (Amended) The expression vector of claim 1, wherein said chemokine gene encodes a chemokine that binds to a C-C chemokine 3 receptor.

11. (Amended) The expression vector of claim 1, wherein said chemokine gene encodes a chemokine that binds to a C-C chemokine 1 receptor.

12. (Amended) The expression vector of claim 1, wherein said chemokine gene encodes a chemokine that binds to a CXR4 receptor.

13. (Amended) The expression vector of claim 2, wherein the secreted chemokine is RANTES (Regulated upon Activation, Normal T cell Expressed, and presumably Secreted), MIP-1α (Macrophage Inflammatory Protein-1α), or SDF (stromal cell derived factor-1).

14. (Amended) The expression vector of claim 2, wherein said secreted chemokine binds to a chemokine receptor.

15. (Amended) The expression vector of claim 14, wherein one or more amino acids are deleted from the N-terminus of the secreted chemokine.

16. (Amended) The expression vector of claim 1, wherein said intracellular retention signal sequence directs a protein expressed from said single intrakine transcript to the endoplasmic reticulum, Golgi apparatus, a lysosome, an intracellular vesicle or other cellular compartment.

17. (Amended) A method of inhibiting phenotypic expression of a chemokine receptor in a cell, wherein the method comprises blocking cell surface expression of said chemokine receptor by binding of said chemokine receptor with an intrakine.

18. The method of claim 17, further defined as comprising the steps of:

obtaining a vector comprising a nucleic acid segment encoding a promoter, an intracellular retention signal sequence and a chemokine receptor binding polypeptide gene; and

transducing said vector into said cell;

wherein said vector expresses said intracellular retention signal sequence and chemokine receptor binding polypeptide gene under the transcriptional control of said promoter to produce a fusion polypeptide when transduced into said cell.

19. The method of claim 18, wherein said polypeptide is a chemokine, a chemokine analog, an antibody or a peptide.

20. The method of claim 19, wherein said polypeptide is a chemokine.

21. The method of claim 18, wherein said polypeptide is RANTES, MIP-1α, SDF, HIV gp120 or the V3 region of HIV gp120.

22. The method of claim 20, wherein said chemokine is RANTES, MIP-1α or SDF.

23. (Amended) A method of inhibiting HIV infection of a cell, said method comprising phenotypically knocking out an HIV co-receptor in said cell by binding of said HIV co-receptor with an intrakine, wherein said phenotypic knock-out of said HIV co-receptor in said cell inhibits infection of said cell.

24. (Amended) The method of claim 23, wherein said co-receptor is a C-C chemokine 5 receptor, a C-C chemokine 3 receptor, a C-C chemokine 1 receptor or a CXR4 receptor.

29. The method of claim 24, wherein said cell is transduced with a CC-chemokine gene fused to an endoplasmic reticulum (ER)-retention signal to intracellularly block the transport and surface expression of an endogenous CC receptor.

33. (Amended) The method of claim 29, wherein said CC receptor is a C-C chemokine 5 receptor (CCR5), a C-C chemokine 3 receptor (CCR3), or a C-C chemokine 1 receptor (CCR1).

34. The method of claim 24, wherein said cell is transduced with a CXC-chemokine gene fused to an endoplasmic reticulum (ER)-retention signal to intracellularly block the transport and surface expression of an endogenous CXR4 receptor.

35. (Amended) An expression vector for treatment of an HIV infection in a subject, wherein said expression vector includes:

an expression region which comprises:

a promoter;

an intracellular retention signal sequence encoding region; and

a chemokine encoding gene;

wherein said intracellular retention signal sequence and said chemokine encoding gene are expressed as a single intrakine transcript from said promoter; and

wherein when said expression vector is administered to lymphocytes, monocytes, macrophages or stem cells of said subject said cells exhibit a phenotypic knock out of an HIV co-receptor.

36. The expression vector of claim 35, wherein said cells are transduced *ex vivo* with said vector.

37. The expression vector of claim 36, wherein said stem cells are autologous stem cells.

38. (Amended) A composition comprising the expression vector of claim 35 and a pharmaceutically acceptable solution.

39. (Amended) A method of increasing white blood cell count in a subject with an HIV infection comprising administering to said subject a pharmaceutical composition comprising lymphocytes, monocytes, macrophages or stem cells transduced with a vector of claim 1, thereby increasing white blood cell count in said subject with an HIV infection.